Iranian Journal of Clinical Infectious Diseases
2008;3(1):39-42

CASE REPORT

Disseminated tuberculosis presenting as hypersplenism

Mansoureh Momen Heravi, Mahmood Khanbanpoor
Department of Infectious Diseases, Kashan University of Medical Sciences, Kashan, Iran

ABSTRACT

Background: Occurrence of hypersplenism in TB is very rare. We report a case of DTB with hypersplenism presentation.

Case presentation: A 23year old man was admitted with complaints of fever, night sweats, anorexia, headache, shortness of breath, early satiety that had started 10 days ago. He had been well until 9 months ago, when early satiety developed for the first time. Physical and ultrasonographic examinations of the abdomen revealed huge splenomegaly. The Results of his blood tests showed pancytopenia Abdominal CT scan revealed a mild hepatomegaly, huge splenomegaly, para-aortic, retroperitoneal, and left inguinal lymphadenopathy. Splenectomy was performed. Pathologic examination of his liver, spleen and lymph node biopsy specimens revealed caseous necrosis and granuloma formation due to TB. After initiating the anti-tuberculosis treatment, all of his signs and symptoms disappeared. At the present time, his general condition is good and he has no any problem

Conclusion: Huge splenomegaly and hypersplenism can occur during disseminated tuberculosis. DTB can mimic lymphoma and it must be considered in differential diagnosis of huge spleen.

Keywords: *Disseminated tuberculosis, Hypersplenism, Pancytopenia.*
(Iranian Journal of Clinical Infectious Diseases 2008;3(1):39-42).

INTRODUCTION

Disseminated Tuberculosis (DTB) refers to the concurrent involvement of at least two non-adjacent organs or sites of the body or, involvement of the blood or bone marrow by tuberculosis process. Splenic invasion in TB occurs in either disseminated form or miliray tuberculosis (1).Occurrence of hypersplenism in TB is rare .We report a case of DTB who presented with hypersplenism.

CASE PRESENTATION

A Twenty three-year old man who was a citizen of Kashan city, was admitted with complaints of fever, night sweats, anorexia, headache, shortness of breath, and early satiety, started 10 days ago. He had been healthy until 9 months ago, when early satiety developed for the first time. His outpatient work up started few weeks before admission.

Physical examinations revealed huge splenomegaly. The peak of fever was at nights however he was afebrile at mornings.

There was no history of underlying or familial disease and also no history of drug use .

Laboratory data revealed white blood cell count of 2130/mm³ (PMN: 54%, Lymph: 44.8%),Platelet count of 84000/µl, hematocrit level of 38.6%, hemoglobin level of 12.6 g/dL, and erythrocyte sedimentation rate of 20 mm/h. PPD test was negative.

Received: 9 December 2006 *Accepted*: 22 December 2007
Reprint or Correspondence: Mansoureh Momen Heravi, MD. Kashan University of Medical Sciences, Kashan, Iran
E-mail: mansoreheravi@yahoo.com

A chest radiograph was obtained and it revealed no abnormal findings. Blood, urine, and stool cultures were performed and all of them were negative.

Transverse ultrasonography of the abdomen reported huge spleen with normal echogenicity and without any mass lesion.

CT images of the abdomen obtained with intravenously administered contrast material revealed a mild hepatomegaly, huge splenomegaly, para-aortic, retroperitoneal, and left inguinal lymphadenopathies.

He lost about 8 Kg of his weight during his outpatient work up so he was admitted for further diagnostic evaluations.

The results of laboratory examinations at the time of admission were:

Alkaline Phosphatase =51 U/L, LDH=2150 IU/L, Total Bilirubin=0.6 mg/dL, Direct Bilirubin=0.2 mg/dL, Ca=10.2mg/dL, BUN=14 mg/dL, Creatinin=1 mg/dL, HBsAg=negative, Urinalysis=normal, Stool-exam=normal, wright & Coombs wright test=negative

WBC= 2000/mm³ (PMN=46%, Lym=51%, Mon=1%, Eos=2%, Hb=11gr/dL, HCT=35.4%, MCV=73fl, MCH=23 pg, MCHC=31g/dL, PLT=80000/µl

CT scan of the chest, smear and culture of sputum and Bone marrow biopsy were obtained. CT scan of the chest showed a few small para-tracheal and para-esophageal lymph nodes with normal pulmonary parenchyma.

Bone marrow biopsy revealed erythroid hyperplasia, microblastic reaction and mild eosinophilia. Smear and culture of sputum for acid-fast bacilli were negative for 3 times.

Regarding to the signs of hypersplenism (huge splenomegaly, pancytopenia), prolonged fever, sweating and weight loss, para-aortic and retroperitoneal lymphadenopathies, a surgical approach was proposed and then splenectomy was performed to rule out lymphoma. Specimens of liver, spleen and lymph node were obtained.

Pathologic examination of his liver, spleen and lymph node biopsy specimens revealed caseous necrosis and granuloma formation including Langhans giant cells, epithelioid cells, lymphocytic and plasma cell infiltration compatible with tuberculosis. Ziehl-Neelsen staining of specimens revealed no acid fast bacilli.

Finally, he was classified as a tuberculosis patient, and anti-tuberculosis treatment with Isonizid, Rifampin, Pirazinamide, and Ethambutol was initiated. Early satiety was the first symptom which improved immediately after splenectomy. His general condition improved 10 days after splenectomy. He was discharged from the hospital and gradually with continuing anti-tuberculosis treatment, all his signs and symptoms resolved, and at last he gained weight .After a few months the CBC result was as the following:

WBC=9850/µl, Hb=14.7g/dL, Hct=43.4%, MCV=78.7fl, MCH=26.6 pg, MCHC=33.8 g/dL, PLT=340000/µl, ESR=14

At the present time, he is doing well and has no problem.

DISCUSSION

Chronic organ tuberculosis is probably always associated with intermittent non progressive seeding of the blood stream. In some individuals however especially as age or other factors compromise immunity, this becomes continuous and produces progressive hematogenous tuberculosis long after the primary infection. The foci responsible for late generalized tuberculosis (LGT) are often clinically silent for example renal, genitourinary, osseous or visceral lymph node. More than one seeding focus is usually present suggesting a change in immune status that favors simultaneously reactivation (2).

Patients with disseminated TB may have splenic involvement, but splenic TB is rarely the main feature in patients with disseminated TB. Splenic TB has been reported in immunocompetent patients

(3-6) But it is more commonly associated with patients infected with HIV and it manifests as splenomegaly or hepatosplenomegaly. Simultaneous involvement of peripheral lymph nodes may occur. The size of spleen becomes normal after TB treatment (7-10). Splenomegaly in TB patients can occur with hematological disorders such as pancytopaenia, myelodisplasia, acute leukemia and chronic myeloid leukemia (11). It's thought that splenomegaly causes hematologic disorders in some patients because they disappear after splenectomy (12, 13).

In our patient, involvement of spleen was due to DTB because of the presence of criterions.

Diagnostic criterions of DTB are as follows:

1-Clinical feature compatible with tuberculosis.

2-Concurrent involvement of at least two non-adjacent organs or sites of the body, or detection of mycobacterium tuberculosis in the blood or bone marrow.

3-Microbiologic and /or histologic evidences of tuberculosis.

4-Significant improvement with anti-TB treatment (1).

However examination for immune deficiency must be considered in this case because Immuno-suppression especially arising from aging is the major cause of LGT (14) but our patient is a young man.

Salvin RE studied 100 cases of LGT with simultaneous reactivation of anatomically unrelated foci in multiple organs and lymph nodes occurred in 54% of cases and 97% of patients exhibited granulomata in Liver biopsy (15).

Huang LM also reported a previously healthy 15-year-old boy who presented with body weight loss, prolonged fever, neck lymphadenopathy, pancytopenia, and hepatosplenic microabscesses and microscopic examinations of both the liver and spleen showed mycobacteria-related granulomatous inflammation and caseating necrosis (16).

Cassim KM reported a 38-year-old black male with a rare combination of disseminated tuberculosis together with a reactive histiocytic haemophagocytic syndrome and tuberculosis hypersplenism which did not resolve despite adequate anti-tuberculosis chemotherapy and splenectomy (17).

Bora P reported a case of 9-year-old girl with a 5-6-month history of abdominal distension and fever who was found to have massive splenomegaly with features of hypersplenism. Splenectomy was carried out and histopathological examination of the spleen revealed granulomatous lesions suggestive of tuberculosis (18).

In conclusion, splenomegaly and hypersplenism can occur during disseminated tuberculosis. DTB can mimic lymphoma and it must be considered in differential diagnosis of a huge spleen.

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